THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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K. G. Parker and Walter H. Burkholder describe the effects of Pseudomonas syringae on apple and pear trees in New York, page 100.

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PEANUT SEED TREATMENTS

Coyt Wilson

The scarcity of labor during the last war and the sharp rise in peanut acreage accelerated the change-over from use of hand-shelled to machine-shelled seed. The rapidity with which this change was made contributed to the troubles that were encountered. The lack of technological information forced operators of peanut shellers to become experimenters. By trial and error, quite a few of them learned how to prepare good seed for planting. However, complaints are still heard occasionally that poor seed have been the cause of poor stands.

Within the last 10 years, experiment stations in peanut-growing States have aided in the improvement of peanut culture by testing many seed protectants under a variety of conditions. However, reports of many of these investigations have been in the form of news releases, or mimeographed recommendations by extension workers, or by short paragraphs in annual reports by experiment stations. Most of the reports are incomplete with respect to methods of applying the protectants, time of applications, storage conditions between time of treatment and time of planting, and the method of determining germination or emergence.

HISTORICAL

Experiments with chemical seed protectants on peanuts were started between 1935 and 1940. One of the earliest official reports was from Australia by Morwood (13) in 1938, who stated that the use of mercurial treatments, Ceresan or Agrosan, at the rate of 5 ounces per 100 pounds of seed, improved the emergence of machine-shelled Virginia Bunch peanuts. Emergence data were not given. The North Carolina Experiment Station reported in 1941 (14) that seed treatment was beneficial on peanuts. According to Shaw (19) this work was started in 1939. Experiments on this problem were started in Virginia in 1939 (25). Although the results of the preliminary work were not conclusive later experiments definitely proved the value of seed treatment.

Most of the information now available on peanut seed treatment has been released since 1942. Shaw (18) reported results of a test made in 1941 with organic sulfur, organic mercury, Spergon, and 20-80 lime-sulfur. Emergence of untreated seed was 23.2 percent. All of the treatments increased emergence. The best treatment was organic sulfur, with an emergence of 65.5 percent. Most of the increase was due to prevention of seed rot within three or four days after planting. In 1943 Shaw (19) stated that New Improved Ceresan, Spergon, Arasan, and Yellow Cuprocide improved stands. Seed treatment was more effective on some seed lots than on others. Machine-shelled seed responded more to treatment than hand-shelled seed.

In 1943 workers at the North Carolina Experiment Station (15) treated hand and machine-shelled Virginia Bunch peanuts 3, 30, 60, and 81 days before planting. There was very little difference in emergence, and it was concluded that seed could be shelled, treated, and stored for "any reasonable period of time before planting."

The Georgia Agricultural Experiment Station reported in 1944 (6) that 2% Ceresan, Arasan, and Spergon consistently improved the stands of Spanish peanuts. Ceresan was best and Spergon was poorest. Treatment had no effect on the yield of individual plants, but it increased yields by increasing the number of plants per acre. Machine-shelled seed after treatment were practically as good as hand-shelled seed.

Effect on emergence of the length of time elapsing between shelling and planting has been studied by the Georgia Station (7). A uniform lot of runner seed was divided into two portions. One portion was shelled in January. These shelled seed were stored with and without treatment until planting time in April. The other portion was shelled and treated just before planting. Treatments used were 2% Ceresan, Arasan, Spergon, Phygon, Dow 9B, and Ceresan M. The seed shelled in January produced stands as good as those shelled just before planting in April. It made no difference whether the seed were treated at the time of shelling or just before planting. All treatments increased emergence, except Ceresan M which was toxic. The experiment was repeated with essentially the same results on Spanish peanuts.

Vaughan stated in 1944 (23) that Arasan, 2% Ceresan, and Spergon increased emergence of machine-shelled seed by as much as 100 percent in Virginia. Arasan was best and Spergon was poorest. The greatest increases were obtained by treating machine-shelled seed of poor quality.

The first reports from Texas (11, 21) stated that seed treatment did not increase emergence nor improve yields. However, a later report by Langley, Reynolds, and Dunlap (12) summarizing the results of three years work at Stephenville, Texas, showed increases of over 600 per-

cent as a result of treating the seed with 2% Ceresan. Arasan and Spergon, although less effective than Ceresan, consistently increased stands. There was very little difference between the emergence of seed treated with 2 or 4 ounces of Arasan per 100 pounds of seed. Increasing the dosage of Spergon from 3 to 5 ounces per 100 pounds of seed did not materially increase emergence.

In 1945 Tisdale (22) reported that Arasan, 2% Ceresan, and Spergon increased germination of machine-shelled seed 25 to 32 percent over non-treated seed. No one of the materials was consistently better than the others.

Seed treatments of peanuts was recommended in Rhodesia in 1945 by Hopkins (9). Proprietary mercurial dusts at the rate of 5 ounces to 180 pounds of seed were recommended as a control for seed rot caused by soil-inhabiting fungi.

Tests made at the Georgia Coastal Plain Experiment Station in 1945 showed that seed shelled and treated in January emerged as well when planted in April as those seed shelled and treated just before planting (8). The rank of the materials, in decreasing effectiveness, was: 2% Ceresan, Arasan, Spergon, Phygon, Dow 9B, and Ceresan M.

Nusbaum (16) tested Arasan, 2% Ceresan, Spergon, and Yellow Cuprocide on unshelled, hand-shelled, and machine-shelled Spanish peanuts at the Edisto Station in 1943. Each material increased emergence on each type of seed. Greatest increases were obtained with machine-shelled seed. The best emergence was from seed treated with 2% Ceresan.

Fenne, in 1945 (3) and in 1946 (4, 5), released the results of a regional peanut seed treatment test carried out in several States. In general, the data confirmed those reported earlier. The possibility of selective action against microorganisms by the fungicides was indicated by the fact that in 1944 at Davis, California, Arasan was the poorest of the treatments, while at Jacksonville, Texas, it was at the top of the list. In 1945, Spergon was second in Alabama but seventh in South Carolina. In 1946, Dow 9B was first in Virginia and seventh in North Carolina.

Results of tests started in Alabama in 1943 have been summarized by Wilson (24). Ceresan M at the rate of 1-1/2 ounces per 100 pounds of seed was found to be better than Arasan, Spergon, Phygon, Dow 9B, Yellow Cuprocide, or Merc-O-Dust. Treatment was most beneficial on machine-shelled seed. Response to treatment on unshelled seed or hand-shelled seed was slight.

At the present time practically 100 percent of the peanuts planted in the Southeast are treated. The practice has not solved all the seed problems, for poor stands still occur at times. Possibly the treatment fails to prevent seed rot under some conditions. Ivanoff (10) in 1947 reported results of tests made in Mississippi where treatment of shelled Spanish seed with Arasan failed to improve stands.

MATERIALS AND METHODS

Unless otherwise stated all tests were made with runner peanuts. The machine-shelled seed were prepared in the U. S. Department of Agriculture sheller described by Reed and Jezek (17), which produces a sample containing a few unshelled seed and about 5 percent splits. One-pound lots and the appropriate amount of the seed protectant were shaken together in paper bags for approximately one minute. The treated seed were then placed on a screen and all splits, unshelled, and moldy seed were removed by hand. Lots of 100 seeds were picked at random from this cleaned sample and hand planted in one-row plots of 20 to 25 feet in length. Usually each treatment was replicated four times in a randomized complete block design; in a few tests there were six replications. Except for those experiments in which storage was a factor, the seed were treated and planted immediately after shelling.

In Table 1 are listed the trade names, the active ingredient, and the standard dosage of the seed protectants tested 2 years or more in Alabama. Several others have been screened out as a result of one year's work.

EXPERIMENTAL

There is an opinion prevalent among peanut growers that seed deteriorate rapidly after shelling. For this reason they insist that seed not be shelled until just a few days before planting time. In 1944 and in 1945, experiments were made to determine the effect of date of shelling on quality of seed. A uniform lot of peanuts was selected and divided into four aliquots. The first lot was shelled about the middle of February and the other aliquots were shelled at 3-week intervals until about the middle of April, when the last shelling was done. All seed were planted immediately after the last shelling date. The results are shown in Figure 1. Seed shelled 9 weeks ahead of planting germinated as well as those shelled one day before planting.

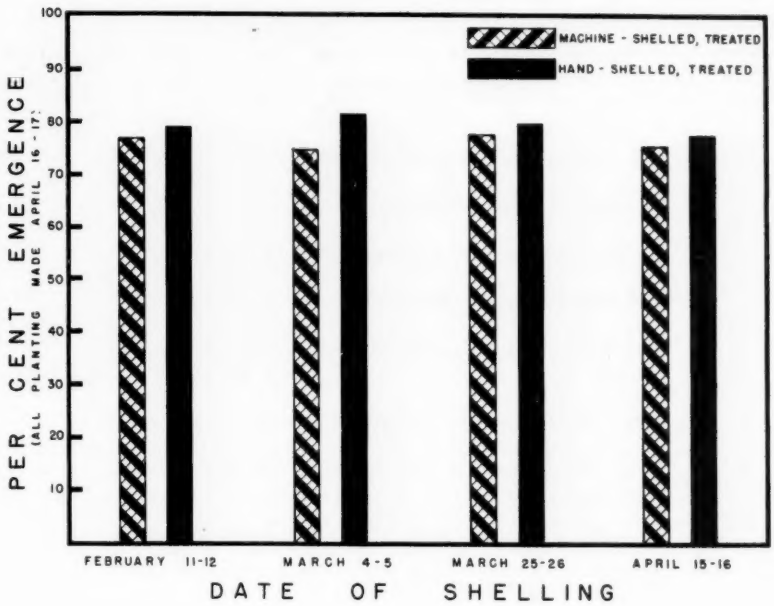

Figure 1. Emergence of runner peanuts shelled 9 weeks, 6 weeks, 3 weeks, and one day before planting. Auburn, Alabama, 1944 and 1945.

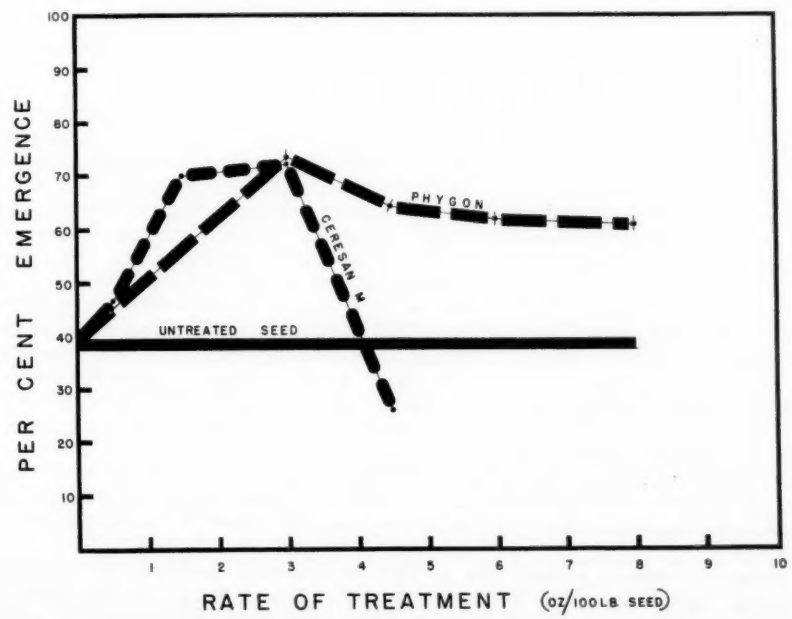

Figure 2. Effect of varying the dosage of Ceresan M and of Phygon upon emergence of machine-shelled runner peanuts, Auburn, Alabama.

Table 1. Trade name, active ingredient, and standard dosage of seed protectants tested two years or more on peanuts in Alabama 1944-1949.

Trade name	Active ingredient	Standard dosage per 100 lb. seed
		oz.
Arasan	50% Tetramethyl thiuramdisulfide	3
Ceresan, 2%	2% Ethyl-mercury chloride	4
Ceresan M	7.7% Ethyl-mercury p-toluene sulfonanilide	1.5
Spergon	98% Tetrochloro para benzoquinone	3
Spergon W	48% Tetrachloro para benzoquinone plus wetting agent	3
Phygon	90% 2, 3-Dichloro-1, 4-Naphthoquinone	3
Phygon W	50% 2, 3-Dichloro-1, 4-Naphthoquinone plus wetting agent	3
Yellow Cuprocide	Yellow cuprous oxide containing 47% metallic copper	4
Vitron D	95% electrolytic cuprous oxide	16
Dow 9B	50% Zinc trichlorophenate	3
Merc-O-Dust	Composition indefinite; contains mercury compounds and formaldehyde	3
Seedox	50% 2, 4, 5-Trichlorophenyl acetate	3

This is in agreement with the data reported by the North Carolina Experiment Station in 1943 and by the Georgia Coastal Plain Experiment Station (8) in 1946. If proper storage conditions are provided, planting seed can be shelled two or three months ahead of planting time.

Several factors enter into the selection of a seed protectant for a particular seed. One of the most important factors affecting choice of a seed protectant is permissible latitude in dosage. Figure 2 shows the effect of varying dosages of Ceresan M and of Phygon upon emergence of machine-shelled runner peanuts. As the dosage of Ceresan M increases, the emergence increases, reaching a peak at 1-1/2 to 3 ounces per 100 pounds of seed. At 4-1/2 ounces per 100 pounds of seed the emergence is less than that of untreated seed. Occasionally, Ceresan M is toxic to seed even at 3 ounces per 100 pounds of seed. A similar curve could be plotted for New Improved Ceresan. As the dosage of Phygon is increased, the emergence increases, reaching a peak at about 3 ounces per 100 pounds of seed. As the dosage is increased above this point, the emergence drops slightly. Even at 8 ounces per 100 pounds of seed, however, the emergence is much better than that of untreated seed. Similar curves could be plotted for Spergon, Spergon W, Phygon W, Arasan, and Dow 9B.

Processors often have to depend upon inefficient help. The protectants are applied with machines that become inaccurate with age or neglect. Under actual working conditions, the dosage applied varies considerably. For these reasons, the Ceresan treatments are not recommended in Alabama.

The nature of mercury toxicity has not been established. Seed that have been overtreated with one of the Ceresan products start to germinate. The primary root may grow out for an inch or slightly more, but the epicotyl does not continue to develop. The hypocotyl continues to increase in diameter and the result is a deformed seedling similar to those shown in Figure 3.

Most of the organic seed protectants mentioned here have been tested at two or more dosage levels. Under the conditions of these tests, there was not a great deal of difference between

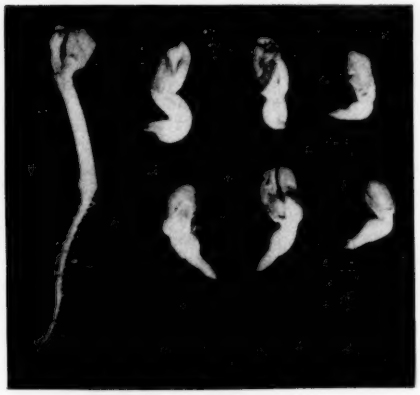

Figure 3. Mercury damage on germinating runner peanuts. The seed at the right were treated with an overdose of New Improved Ceresan. The seedling at the left is normal.

dosages of 2, 3, or 4 ounces per 100 pounds of seed. The results of 2 years work with Arasan, Spergon, and Dow 9B are given in Table 2. The data are in agreement with those obtained from similar tests with one or more of the materials in other years. Even though dosages of 2 ounces per 100 pounds have been found to be as efficient as 3 ounces per 100 pounds of seed for Arasan and Dow 9B, larger doses usually are recommended because of the safety factor. If the dusting machine is set to treat at 3 ounces per 100 pounds of seed, a 33 percent variation is not harmful; however, the same variation would result in under treatment for some seed if the machine were set to treat at the rate of 2 ounces per 100 pounds of seed. An attempt has been made to eliminate this danger in Spergon W by reducing the concentration of the effective ingredient so that the dosage may be kept at 3 ounces per 100 pounds of seed and the cost reduced.

Table 2. The effect of dosage upon the effectiveness of Arasan, Spergon, and Dow 9B.

Treatment	Dosage per 100 lb. seed	Increase in emergence due to treatment		
		1947	1948	Mean
	Oz.	Percent	Percent	Percent
Arasan	2	17.5	49.0	33.3
	3	16.6	55.3	35.9
Dow 9B	1	3.9	1.6	2.7
	2	6.4	33.0	19.7
	3	12.5	22.3	17.4
Spergon	3	7.0	27.0	17.0
Spergon W	3[1]	4.6	25.5	15.0

[1] Equivalent to reducing the dosage of Spergon to 1.47 ounces per 100 pounds of seed.

The regional peanut seed treatment tests reported by Fenne (3, 4, 5) showed that the response to a given seed protectant varied with the location. One of the best materials at one location may be one of the poorest at another location. The reason for this has not been established but probably is related to variations in soil and climate at the time of planting. The same kind of variation in efficiency of seed protectants can be demonstrated by comparing the same materials over a period of years at the same location. Figure 4 shows the relative value of Dow 9B, Spergon, Arasan, and Ceresan M in the years 1945 to 1949. The data are shown as percentages increase in emergence over the untreated check. The data were calculated by the following formula:

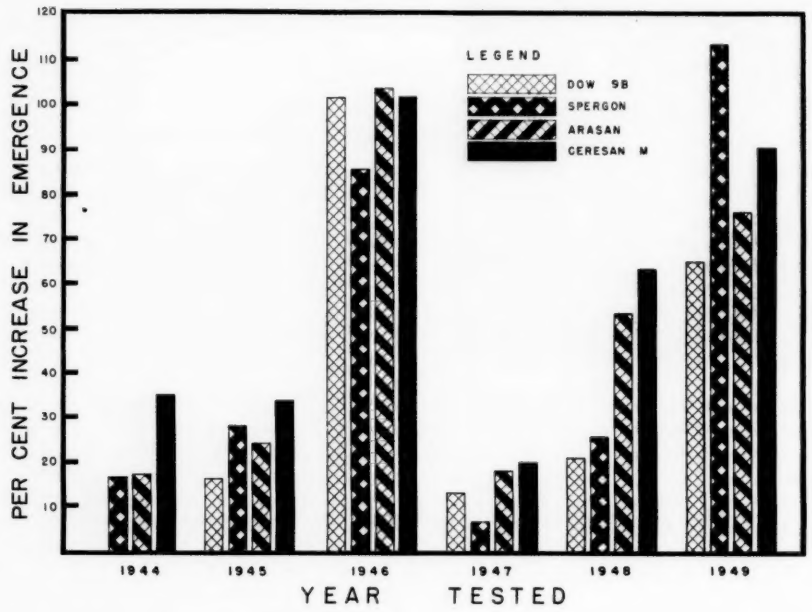

Figure 4. Relative efficiency of Dow 9B, Spergon, Arasan, and Ceresan M on emergence of machine-shelled runner peanuts in 6 different years at Auburn, Alabama.

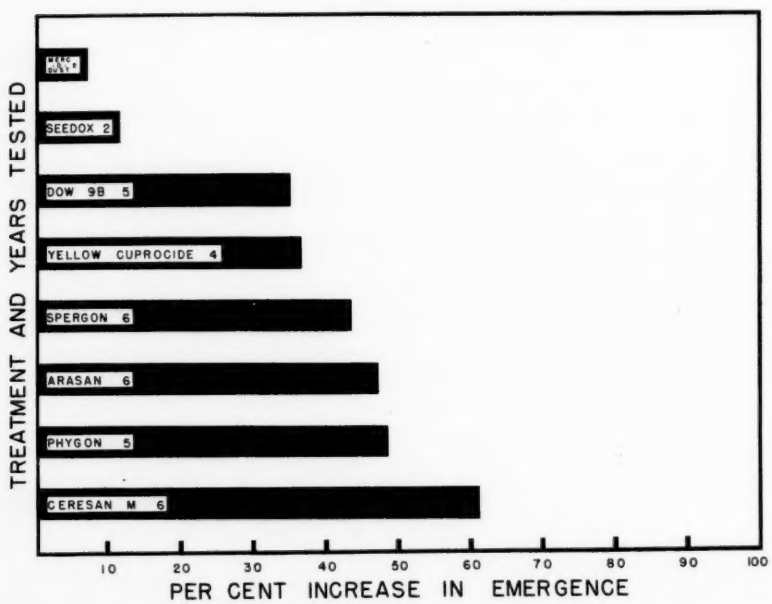

Figure 5. Relative efficiency of eight seed protectants on machine-shelled peanuts, Auburn, Alabama. Tests made during the period 1944-1949.

$$\text{Percent increase in emergence} = \frac{\text{Emergence of treated plot - emergence of untreated plot}}{\text{Emergence of untreated plot}} \times 100$$

Dow 9B was not in the tests in 1944 and for that year only Ceresan M. Arasan, and Spergon are shown. In 1947, Spergon was the poorest of the four treatments. In 1949, Spergon was the best of the four, while in 1946 Dow 9B was as good as Arasan or Ceresan M. On the other hand, in 1948 Dow 9B and Spergon were poor treatments in comparison to Arasan and Ceresan M. Of the four materials, Ceresan M was the most consistent during the six-year period. Arasan was almost as consistent as Ceresan M.

During this six-year period, the average percentage of increase in emergence for the four treatments varied from a low of 14.1 in 1947 to a high of 98.1 in 1946. Climatic conditions were unfavorable for germination in 1946 and in 1949. Under these unfavorable conditions, the protection offered by treatment is reflected by greater increases in stands. When climatic conditions are more favorable for germination, the need for protection is less and the increases in emergence are not so great.

During the six-year period 1944-1949, about 20 materials were tested on runner peanuts. Some of these were in the tests every year, while others were included in only a part of the period. This is due to the fact that some materials made such a poor showing the first year or two that they were discontinued, while some of the others were not available until recently.

Emergence of untreated seed in these tests has varied from less than 15 percent to more than 75 percent, indicating the important effect of external environmental factors and quality of seed on germination and efficiency of the treatment. This variation makes it difficult to compare emergence data from tests made in different years. Therefore, they have been summarized and presented as a bar graph in Figure 5. These data are for machine-shelled runner peanuts, except for 1944 when machine-shelled Spanish seed were used. The data as shown are percentages of increase in emergence calculated as explained previously. In general, the treatments fall into three groups. There is a "poor group" that usually increased the emergence less than 15 percent. Merc-O-Dust and Seedox are in this group. The "good group" usually increased the emergence 30 to 50 percent. Included in this group are the copper compounds, the quinone compounds, and the organic sulfur compounds. The "excellent group" usually increased the emergence more than 50 percent. It is illustrated by Ceresan M. At equivalent dosages, 2% Ceresan and New Improved Ceresan were equally good.

DISCUSSION

Mechanical peanut shellers damage a high percentage of the seed. When the seed are passed over shaking screens, most of the split seed and the undersized seed are removed. The remaining seed pass over a moving canvas belt and most of the visibly damaged seed are removed by hand picking. The finished product, however, contains many seed with broken seed coats, small chipped spots, and minute cracks. When these seed are planted, they are much more susceptible to attack by soil-borne fungi than undamaged seed.

The seed also carry inoculum of saprophytic or weakly pathogenic fungi, such as species of Fusarium, Sclerotium, Alternaria, Curvularia, Basisporium, and Rhizoctonia, according to Crosier (1) and to Evans and Poole (2). By use of seed protectants, invasion of the seed by microorganisms is retarded. The value of the treatment apparently is limited to its protective action during the time the seed is in the soil. No evidence has been presented to show that seed treatment controls post-emergence damping off of peanuts.

Although there have been reports that peanut seed may carry inoculum of Cercospora personata, Sclerotium rolfsii, and vascular wilt fungi (20), there is no evidence that seed-borne inoculum of these fungi play an important role in reduction of peanut stands. It is an established fact that seed treatment does not satisfactorily control any of these diseases.

That the fungicide weathers away very quickly after the seed are planted is evident from the fact that no instance has ever been reported where treatment interfered with inoculation.

It has not been possible to show by any of the tests conducted in Alabama that any of the protectants used hastens germination or increases the vigor of the plants. In some tests yield records were obtained, but there was no evidence that the yield per plant was influenced by the use of any of the protectants.

All the available evidence at present indicates that the only value of the protectant is the prevention of seed rot after planting which in turn results in better stands.

SUMMARY

1. During the 6-year period 1944 to 1949, most of the commercially available seed protectants were tested for their value in improving the emergence of runner peanuts.
2. The need for seed treatment is much greater with machine-shelled seed than with hand-shelled seed.
3. Machine-shelled seed are practically as good as hand-shelled seed if they are treated with a suitable seed protectant prior to planting.
4. The best emergence usually was obtained from seed treated with 2% Ceresan or Ceresan M.
5. Over-dosage with mercurial treatments decreases emergence below that of untreated seed. Treatment of seed with more than the recommended dosage of Dow 9B, Spergon, Arasan, or Phygon did not have any appreciable ill effect on the seed.
6. The response to seed treatment varied with varying climatic conditions. Seed treatment was very effective in 1946 and in 1949. In 1947, treatment did not increase emergence appreciably.
7. Specificity of fungicides was evident from the fact that in 1947 Spergon was less effective than Dow 9B, Arasan, or Ceresan M, but in 1949 it was more effective than any of these. A similar trend was evident with Dow 9B.
8. Seed shelled and treated the middle of February germinated as well when planted in mid-April as those seed shelled and treated one day before planting.
9. The principal value of seed treatment is in the prevention of seed rot between the time of planting and germination. The value of seed treatment for the control of seed-borne diseases is doubtful.

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ALABAMA POLYTECHNIC INSTITUTE

SOYBEAN DISEASES IN IOWA IN 1949[1]

J. M. Crall

The following diseases occurred on soybean in Iowa in 1949: bacterial pustule (Xanthomonas phaseoli var. sojense), downy mildew (Peronospora manshurica), bud blight (virus), brown spot (Septoria glycines), root and stem rot (Rhizoctonia solani), wildfire (Pseudomonas tabaci), charcoal rot (Macrophomina phaseoli), mosaic (virus), brown stem rot (Cephalosporium gregatum), and stem canker (Diaporthe phaseolorum var. batatatis).[2] Only the latter two diseases were considered to be causing serious losses.

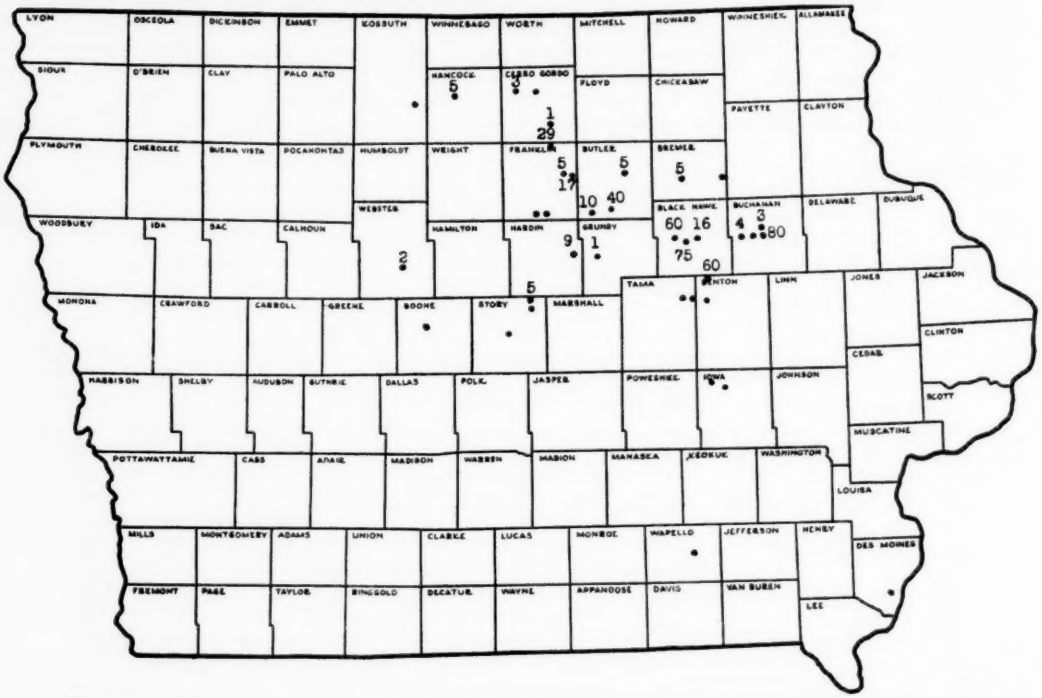

Figure 1. Occurrence of Diaporthe stem canker on soybean in Iowa in 1949. Each dot represents a field in which a trace or more of infection was found. Figures represent per entage of plants infected where infection was one percent or more.

Bacterial pustule was first noted July 5 at Ames. It was found subsequently in almost all fields seen throughout the State. Maximum infection occurred in mid-July. Infection on lines in the breeding plots at Ames varied from a trace to an abundance (with marked defoliation) at that time. Infection on varieties in Uniform Nurseries (Group II and Group III) was recorded as follows: slight - Bavendar Special; slight to moderate - Hawkeye, Earlyana, Richland; moderate - Blackhawk, Chief; abundant - Adams, Lincoln, Dunfield, Illini. There was little further spread of the disease after mid-July, and even severely infected plants made fairly rapid recovery.

Downy mildew was first noted July 5, and bud blight July 21 at Ames. Neither of these diseases was found in more than trace amounts, either at Ames or throughout the State. Brown spot and mosaic were seen in some fields during the season but were not important. Plants

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[1]Journal Paper No. J-1729 of the Iowa Agricultural Experiment Station, Ames, Iowa. Project Number 883.

[2]"Stem canker" is suggested as a descriptive name for the disease of soybean caused by Diaporthe phaseolorum var. batatatis to distinguish it from "pod and stem blight" caused by D. phaseolorum var. sojae. See: A. W. Welch and J. C. Gilman, Hetero- and homo-thallic types of Diaporthe on soybeans, Phytopath. 38: 628-637. 1948.

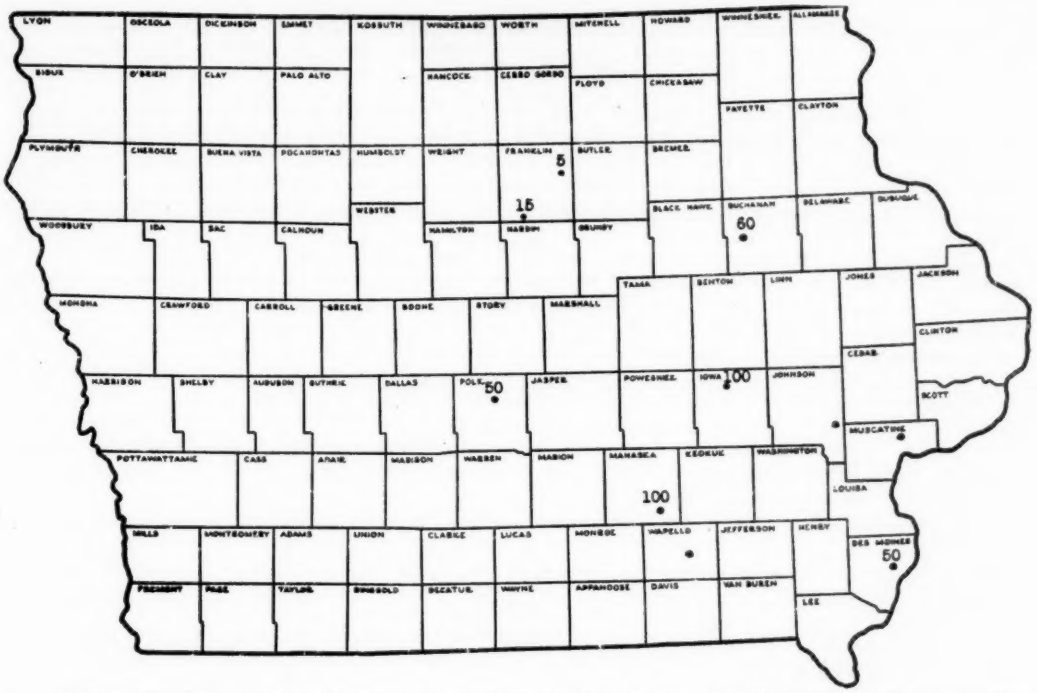

Figure 2. Occurrence of brown stem rot on soybean in Iowa in 1949. Each dot represents a field in which a trace or more of infection was found. Figures represent percentage of plants infected where infection was more than a trace.

with Rhizoctonia root and stem rot were received about August 1 from a field in north central Iowa. Charcoal rot was found in September in one field in central Iowa. Wildfire was found the latter part of August in experimental plots at Ottumwa. It was limited to two ranges at one end of a plot. Although it was not found in commercial plantings, surveys were not adequate to preclude the possibility of its presence there.

Diaporthe stem canker and brown stem rot were the two most important diseases of soybean in Iowa in 1949. Stem canker was first found on August 15, at Ames. It was found later at numerous locations in the State, with the most severe infection in fields in Franklin, Butler, Blackhawk, and Bremer Counties, in an area centered near Waterloo. The accompanying map (Fig. 1) shows the distribution of the disease. Among fields in which infection was severe were four, 20 to 30 acres in size, with 40, 60, 60, and 75 percent of the plants infected. The percent yield losses from Diaporthe stem canker were difficult to estimate accurately. Since the disease occurs late in the season, the pods of early maturing varieties are set and apparently well filled, at the time of appearance of disease symptoms. The pods on the upper portion of diseased plants of late maturing varieties yield few or no seeds. Yield loss was estimated as at least 10 percent in one field. Counts of plants with stem canker were made in experimental plots at Hudson (Blackhawk County) on September 13. Percentages of infected plants based on counts of 300 or more plants per variety were: Bavendar Special - 5; Lincoln - 15; Adams - 22; Illini - 18; Dunfield - 28.

Brown stem rot was found in mid-September at various locations in central and eastern Iowa Distribution of the known occurrences of this disease is shown in an accompanying map (Fig. 2). Leaf symptoms of the disease were uncommon. As a result, the disease was probably not found in numerous additional fields in which it occurred. Among fields in which infection was severe were two 30-acre fields with 100 percent of the plants infected.

DIVISION OF FORAGE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, AND IOWA AGRICULTURAL EXPERIMENT STATION, AMES, IOWA

CORN DISEASES IN INDIANA IN 1949

Arnold J. Ullstrup

The general increase in prevalence and severity of the more common corn diseases, and the occurrence of a few unusual diseases of this crop seems worthy of comment.

Seedling blight in late May and June was more prevalent than has been observed in several years. Some of the most severely affected stands had been planted with untreated, mechanically damaged, single cross seed. Isolations from infected seedlings indicated that Penicillium oxalicum was at least partially involved.

Pythium stalk rot became prevalent during the first part of July following a period of hot, humid weather. The disease was most destructive in bottom lands in southern Indiana, although one field in the northern part of the State showed 10 percent of the plants infected. Isolation and reinoculation, in parallel series of P. butleri and a number of cultures of bacteria, confirmed the virulence of the fungus. None of the isolates of bacteria caused infection on reinoculation.

Brown spot disease caused by Physoderma zeae-maydis appeared to be unusually abundant in southern Indiana where considerable infection was observed on the leaf blades. The disease, however, caused only a minor amount of stalk infection. One isolated case of this disease was found as far north as Lafayette. The check in the spread and severity of the disease following abundant early development was probably due to the relatively low mean temperatures and reduced rainfall in August and September.

"Crazy Top" of corn was relatively more abundant than has been seen in the past several years. In all instances the disease occurred in low areas in fields where flooding or water-logging occurred shortly after planting. Studies on this disease have indicated that the causal organism is Sclerospora macrospora. Experiments with seed harvested from infected plants have shown that seed transmission of the disease is possible. From a practical standpoint this is unimportant since a large percentage of infected plants are barren, and when not, produce only small nubbin ears that are likely to be missed by mechanical pickers and certainly discarded during the processing of seed.

Rust caused by Puccinia polysora was found for the first time in Indiana along the Ohio River. This species, generally confined to tropical and sub-tropical regions, occurred only sparingly. Specific identification was confirmed by Dr. George B. Cummins.

Bacterial leaf blight caused by Pseudomonas alboprecipitans was prevalent on a number of inbred lines and to a lesser extent on hybrids. The identity of the disease was based on symptoms observed in the field, cultural characters of the organism, and symptoms produced on reinoculation of seedlings.

Stewart's disease caused by Bacterium stewarti was moderately prevalent on sweet corn early in the summer, but late infection on dent corn did not develop to any significant degree in 1949.

Northern corn leaf blight caused by Helminthosporium turcicum was present to a greater extent in the southern two-thirds of Indiana than has been seen since 1943. In some localized areas severe epidemics had become established by August 15. The high humidities that prevailed during late July and early August probably accounted for the increase of the disease. Where epidemics developed early (by August 15) considerable reduction in grain yield took place, as judged from the appearance of ears in those fields where the disease was severe.

Southern corn leaf blight caused by H. maydis was prevalent in the southern one-third of the State. This disease appears to be increasing in prevalence in Indiana and has moved northward from the Ohio River bottoms within the past few years.

Stalk rot of corn was present in normal amounts with no indication that the disease was excessively severe except in localized areas where it was accentuated by the leaf blights. Diplodia zeae was responsible for most of the stalk rot, although Gibberella zeae was found in some areas.

Ear rots, with few exceptions, were appreciably less abundant than is normally observed. Diplodia ear rot and gray ear rot caused by Physalospora zeae were particularly less prevalent, only the latter being present to any noticeable degree in one localized area in Bartholomew County. Gibberella ear rot, although relatively more abundant than in a normal year, was not severe; only the extreme tips of ears were affected. There was some slight increase in cob rot caused by Nigrospora oryzae, but no appreciable damage occurred.

The disease situation on corn in Indiana in 1949 appears to be directly correlated with the weather conditions that prevailed during the growing season. Diseases such as Pythium stalk rot, Physoderma disease, bacterial leaf blight, and rust caused by P. polysora, that are generally found in the warmer and more humid corn growing areas such as the Gulf States, probably developed as a result of the abnormally high temperatures and humidities during June and July.

Excessive rains in late May, that occurred on planted fields between onset of germination and when seedlings were four to six inches high, provided the conditions necessary for infection by Sclerospora macrospora. The high humidities in the latter part of July and early August provided favorable conditions for spread and development of the leaf blights caused by Helminthosporium species. The low mean temperatures during the latter part of August and September together with abnormally low rainfall in this period probably were effective in reducing the amount of ear rots.

U. S. DEPARTMENT OF AGRICULTURE AND THE PURDUE UNIVERSITY AGRICULTURAL EXPERIMENT STATION, LAFAYETTE, INDIANA

A WESTERN WHITE PINE TWIG CANKER

Ross W. Davidson

The fungus Curcurbidothis pithyophila was collected on twigs of Pinus monticola near Nakusp, British Columbia, August 29, 1949, and identified by P. L. Lentz of the U. S. Division of Mycology and Disease Survey. This fungus was fruiting on numerous cankered areas of small twigs in the lower crown of one pole-sized tree.

F. Petrak (Ann. Myc. 19: 198-201, 1921) described the fungus from a Weir specimen (10893) collected on Pinus flexilis twigs in Idaho, September 1917. A part of this specimen is in the Mycological Collections, Bureau of Plant Industry, Plant Industry Station, Beltsville, Maryland. A second specimen was collected by E. P. Meinecke and J. S. Boyce on twigs of Pinus monticola in Columbia National Forest (now The Gifford Pinchot National Forest), Washington, in August 1942 (identified by J. S. Boyce and W. W. Diehl). This indicates that it may be widespread in the western white pine area. No information is available regarding its prevalence or pathological importance.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

PSEUDOMONAS SYRINGAE VAN HALL ON APPLE AND PEAR IN NEW YORK STATE

K. G. Parker and Walter H. Burkholder

In 1946, the county agricultural agent, Mr. C. G. Small, in Wayne County, New York reported a very severe blighting of the blossoms in a pear orchard located some four or five miles south of Lake Ontario. When the orchard was visited in mid-summer, most of the blossoms had dropped; but a considerable number of dead blossom clusters were still attached to their spurs, and in some cases one-fourth to one-half inch of the tip of the spur was dead. The Sheldon and Seckel varieties were severely affected, with very few fruits present on the trees; but the Bartlett variety appeared to be only moderately affected. Superficially, these blighted blossoms bore some resemblance to those killed by the fire blight pathogen, but detailed examination clearly revealed differences which are described below.

This disease appeared again the following year in the same orchard but in somewhat less severe form. In 1948 and 1949, it occurred but was of little importance. During these two years similar symptoms occasionally were observed in other pear orchards.

In 1947, in the same county, Mr. Small reported severe blighting of fruit spurs on Wealthy apples in one orchard. On the apples, infection also appeared on the new terminal growths. Lesions on both fruit spurs and terminals were well developed at the time of full bloom, infection apparently having occurred before the blossoms opened. This apple orchard was not visited in 1948, but observations in 1949 again revealed a blighting of a type somewhat different from that caused by the fire blight pathogen. Most of the infections were in the terminal growths and the disease was relatively unimportant in this orchard in 1949. Again similar symptoms have been noted in other apple orchards.

Symptoms

In the field the disease on pear has been observed with certainty only on the blossoms. The receptacle is blackened or very dark brown, as is the pedicel when the lesion extends that low. Extensive invasion does not occur on the trees in which this disease has been encountered but the trees have been in moderately low vigor. In fact, many times only part of the receptacle shows evidence of disease; however, the tips of the fruit spurs sometimes are killed. There is no reddish color in the invaded tissue as is caused by the fire blight pathogen, but in the very early stages watersoaking of the tissue is evident at the advancing margins of the lesions. Occasionally, black necrotic spots appear on the leaves but no study was made to determine whether or not these are caused by the same disease as that described on the blossoms.

On apple, the diseased bark tissue is almost black and the dead blossoms are dark brown in color. Frequently, brown streaks in the outer xylem extend several inches longitudinally beyond any external evidence of infection. No invasion of large branches has been observed, but infection through the terminals may extend downward into the previous years' growth. Cankers have occurred on twigs up to 3/8 inch in diameter. The apple trees on which the disease has been identified were, unlike the pears, in moderately high vigor.

On both apple and pear pronounced swellings develop immediately below the dead part of a fruit spur or twig. Ooze was not associated with any of the lesions.

The appearance of the disease led one to believe that it was of bacterial origin; and microscopical examinations of these blighted pear blossoms and also the infected apple twigs revealed that they contained an abundance of bacteria. Dilution plates made from diseased material in the spring of 1947, 1948 and 1949 yielded in all cases bacterial colonies similar in appearance. Thirteen cultures were saved from various isolations for inoculation experiment and cultural studies. It was readily seen that the organism was not Erwinia amylovora (Burrill) Winslow et al.

Pathogenicity

In August 1947 freshly collected green fruits of Bartlett and Kieffer pear were inoculated with isolates obtained from Sheldon pear blossoms and spurs collected in May of the same year. Small black lesions resulted around the points of inoculation. These consisted of depressed areas 3 to 6 mm. in diameter and 5 to 6 mm. deep. In 1949, Bartlett pear blossoms in the field were inoculated with isolates obtained in 1948 from Sheldon and Seckel blossoms. A bacterial suspension in sterile distilled water was applied to each open blossom by means of a camel's hair brush. Of 82 blossoms persisting 12 days after inoculation with cultures from the Sheldon variety, 53

were diseased, in 21 of which the pedicels were invaded. In 98 blossoms inoculated with isolates from Seckel, 20 showed some evidence of disease. Reisolations from these blossoms yielded cultures identical with those used in making the inoculations. When compared with Erwinia amylovora in artificial inoculations on pear blossoms, lesions appeared in a shorter time but soon ceased development.

In the United States, Wilson (7) and Rosen (6) have reported that Pseudomonas syringae van Hall may cause a blossom blight of pear similar to the one under study. The latter found it in wet cloudy weather during bloom and such was the case in 1946 when first observed in New York State. Clara (3) also described a blight of pear caused by Pseudomonas utiformica but this species is now considered a synonym of Ps. syringae, as is Pseudomonas prunicola reported by Wormald and Montgomery (8) to cause a blossom blight of pear in England. The inoculations made in the present study caused lesions similar to those obtained by Clara with the organism he described. Other bacteria described as pathogenic on pear blossoms are B. barkeri Berridge (2) first reported by Barker and Grove (1) without giving it a name; and Pseudomonas nectrophila Doidge (4). The former was found in England and the latter in South Africa. These two organisms are closely related to Ps. syringae and if comparative studies could be made the three might prove to be identical. Aplanobacter fleuryi Mariat (5) has been reported on pear trees in Switzerland, but this species is distinct from those pathogens listed above, and differences also are noted in the symptoms it produces on the pear.

From the above review of literature it was considered likely that the pathogen with which we were dealing was Ps. syringae. Further experiments, therefore, were conducted to prove or disprove our assumption. First, inoculations were made with the pear and the apple isolates on the stem of young Red Kidney bean plants and on the leaves of lima beans grown in the greenhouse. These two plants are hosts of Ps. syringae and develop rather typical symptoms when infected with this pathogen. In our experiments the symptoms that developed on the beans were the same as have been obtained with known cultures of Ps. syringae.

Second, experiments were conducted to determine the cultural characteristics of the 13 isolates which had been used in the various inoculation experiments. The characters proved to be identical with all cultures. The bacteria produce a water soluble green fluorescent pigment in Clara's medium, liquify gelatin, utilize sucrose, turn milk alkaline, reduce nitrates in nitrites and show a scant to no growth in broth plus 5 percent salt. These characters are the most distinctive of Ps. syringae, and the pathogen therefore is considered to be this species.

Since the disease occurred in the same orchards from its first observation in 1946 until 1949, some attempt was made to discover the source of inoculum. On Long Island, Ps. syringae infects lilac plantings and also Prunus spp. in the fence rows, and from these plants spreads to lima beans where at times it becomes very troublesome. Lilacs were not found in the vinciity of diseased pear and apple orchards although both wild and cultivated cherry were found. Cankers occurred on these trees but attempts to isolate Ps. syringae from them during the summer of 1949 met with failure. There is the possibility, however, that the pathogen may overwinter in the small cankers on apple where lesions are observed on twigs up to 3/8 inch in diameter, but this has not been verified. On pear it seems less likely to overwinter since infections have not been observed to progress more than 1/2 inch down the spur.

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DEPARTMENT OF PLANT PATHOLOGY, CORNELL UNIVERSITY, ITHACA, NEW YORK

PAPERS PRESENTED AT THE PITTSBURGH SYMPOSIUM ON THE TEACHING OF PLANT PATHOLOGY

INTRODUCTION

A symposium on the teaching of plant pathology was a feature of the Fortieth Annual Meeting of the American Phytopathological Society, at Pittsburgh, December 6, 1948. For one reason or another, the five papers presented at that meeting have never been published.

The analytical discussion that characterizes these papers should be a help, not merely to teaching pathologists, but to all plant pathologists, regardless of whether or not they agree with particular viewpoints. For this reason the Plant Disease Survey has agreed to publish them in the Plant Disease Reporter.

Papers read at the symposium dealing with graduate study in plant pathology, during the New York meeting last December, will be published later. -- P. R. M.

DO PLANT PATHOLOGISTS NEED EDUCATION?[1]

Frank D. Kern

The scientific knowledge and technical skills that have made plant pathology possible are the products of education and research. If plants are essential, if their protection is of paramount importance, plant pathologists are indispensable and their education is of supreme consideration. On the day that I begin writing this paper my attention is called to an advertisement in the newspaper, "Education is still the important issue in the Regular Army. Get all the details by calling at your nearest Army recruiting station." But where is education not an important issue? "Education is an institution of every civilized society", the instrumentality of all technical or professional training.

The title of this talk was proposed by the program committee. It is the first of a series of considerations on the teaching of plant pathology. In general the purpose of this Conference is the interchange of experience among teachers of plant pathology. I propose to begin with a discussion of some broad questions such as objectives, goals, and values, as they appear to me. I say this because I have consulted no one and I cannot speak for my colleagues.

Many ancient peoples were distinguished by their esteem for education. The past century, however, has been outstanding in human history witnessing the spread of education to all parts of the world and the increased diversification of education for specialized ends, professions and vocations--even education itself has become a profession. Each of the numerous branches of education has its own objectives but it has been well said that "they possess in common a half-formed faith that there exist certain cardinal principles that we can rely on to guide us in the complicated business of living". It may be pointed out that one meaning of the term profession is "devotion to higher ends than merely earning a livelihood".

Duggar in his book, Fungous Diseases of Plants, published in the year that this society was founded (1909) said that "there is no plant pathology which deserves the name affixed to it prior to the appearance of Kühn's 'Die Krankheiten der Kulturgewächse,' Berlin, 1858." In an Encyclopedia, dated 1912, I find the statement, "Scientific investigation of the diseases of plants has not till recently been so widely and systematically followed up as the economic importance of the subject deserves". Those of us who were active in 1912 are perfectly willing to admit the truth of the next statement to the effect that our knowledge, then, of the causes and of the conditions of disease in the vegetable kingdom was comparatively limited and imperfect. Since that time we have made much progress. Plant pathology has made its way in the colleges and universities as a phase of botanical science or with independent departmental standing. It has been concerned with teaching, research, and extension.

Having come to occupy a place of recognition and standing in our scheme of higher education, plant pathology must be prepared to share some of the criticisms directed toward higher education in these days. Faced with such challenges as that of the Presidents Commission on Higher Education, and many others, most colleges and universities have begun "an intensive reexamina-

[1] A paper presented in The Symposium on The Teaching of Plant Pathology, Fortieth Annual Meeting of The American Phytopathological Society, Pittsburgh, Pennsylvania, December 6, 1948.

tion of their philosophies, their curricula, and their teaching methods." Higher education appears to be in a state of ferment. We cannot leave the problems of our education to others. If we are to have an improved approach to plant pathology education, it must be sought by plant pathologists.

We have plenty of company if we resolve to consider how we may improve our professional education. Representatives of several professions recently held a conference (at Buck Hills Falls, Pennsylvania, April 12-14, 1948) to consider the common problems which education in their fields faces today and to consider means for solving these problems. Their report "Education for Professional Responsibility" (Carnegie Press, 1948) is a most interesting contribution to the type of consideration before us today. They divided their discussions into three parts (1) objectives of professional education (2) the content and methods of professional instruction and (3) humanistic and social education as preparation for professional responsibility and citizenship. I wish everyone of you might read this report. It is a fine presentation of many of today's problems in education.

Without doubt you might be prepared for a certain analogy between plant pathology and medicine, but how about law? In a paper by Professor Lon J. Fuller, of Harvard, the virtues, limitations, and dangers of four competing conceptions of the objectives of legal education are examined. The four conceptions are: (1) The object of legal education is to give the student knowledge, (2) The object is to impart skills, (3) True education consists in exposing the student to great minds, (4) The object of legal education should be to give the student an understanding of, and an insight into, the processes in which the lawyer participates. These four concepts apply in principle to plant pathology. Perhaps most of us would agree that each of these four concepts should be viewed as supplementing the others. So far as knowledge and skills are concerned we would agree with the statement of another speaker that "the objective must be both to transmit professional knowledge and to develop the skill to use it. Knowledge without skill is ineffective, and skill without knowledge is dangerous". The report of the President's Commission on Higher Education for American Democracy does not blame the schools and colleges, solely or even mainly, for the crisis in which the world finds itself but it does say that the knowledge and skills that have made atomic and biologic warfare possible "are the products of education and research, and higher education must share proportionately in the task of forging social and political defenses against obliteration." They speak slightingly of slow superficial curriculum tinkering and plead for measures which will "match in boldness and vision the magnitude of the problem."

Surely we are justified in looking to the institutions of higher education for significant contributions to the maintenance of civilization and the progress of mankind. So much as has been said and written recently about the need for citizenship training that I hesitate to bring up the subject here but I cannot refrain. Perhaps the committee regards the knowledges and skills of the science of plant pathology as training and the social and humanistic aspects of preparation, if any, as education. If that is what is meant by the query "Do Plant Pathologists need Education?", my answer is yes. If the men of our profession are to assume places of democratic leadership, as all professional men should in spite of the demands which their professions make upon their time and energies, they must be better prepared educationally for citizenship than in the past.

I believe that we ought to educate all professional students for citizenship. I wish I could lay before you a definite plan for the achievement of this objective. It has been said that "awareness of a problem is a very important step toward solution." If we come to the realization that we have put comparatively too much emphasis on the professional side and too little on the social side of our educational system, then I believe we will do something about the situation. But some of you may ask whether this be true. I answer with a question--are not the world's needs today social rather than material? Dean Arthur T. Vanderbilt, of New York University, has said, "The salient characteristic of our age is not science or technology or atomic energy. On a world-wide basis it is politics and in many parts of the world power politics. There is no gainsaying this great actuality of modern life. If we are wise, we will recognize it as one of the fundamental premises of our professional education." Must we not agree that it will avail us nothing to turn out "the best professional men imaginable if they have only a very sick society in which to live and work."

To the charge that "Present college programs are not contributing adequately to the quality of student's adult lives either as workers as or citizens," I am afraid we will have to plead guilty. Certainly many prominent educators agree that the college must prepare its students for citizenship since the ivory tower concept of education is gone forever. Not long ago a certain university, upon the occasion of the 100th Anniversary of its founding, decided to honor a

comparatively small group selected as its most distinguished alumni. Ninety-nine were chosen among the 40,000 living alumni. Dr. Daniel Starch, an eminent psychologist, thought this "an unusual opportunity to ascertain what a group of highly selected persons of distinguished achievement considered were the net results of their years in college". Dr. Starch accordingly addressed a set of questions to these persons in an attempt to get them to evaluate their college training. I have the complete report and I know it would interest you in its entirety if time permitted, but we must be content with some conclusions derived from his 72 replies. "Influence of personalities and manner of teaching" tops a list of seven values, almost tied is "training in where and how to acquire further knowledge and skill" and also near the top is "training in ability to think, analyze and organize thoughts". "Actual knowledge acquired in college" is next to the bottom of the list, ranking below "understanding of and dealing with people", and being exceeded in unimportance only by the "influence of fellow students".

When so many academic people rate the "acquiring of knowledge" so high why do these successful persons rate it so low? Dr. Starch believes there are several reasons. He points out first, that students acquire much information merely to pass examinations and it then fades away quickly, and second, that much information gained is trivial or "appears trivial because teachers do not relate it to more important values," and third, that it is impossible anyway to assimilate in a few years in college much of the information related to a profession or business that will necessarily have to be acquired in life. What to do about it? He suggests a re-evaluation of knowledges and facts to be presented in college curricula accompanied by a house cleaning, a "closer integration of the remaining choicer subject matter not merely with making a living but with living itself", and a "vigorous guidance of students in learning not merely to remember but to think." He wouldn't throw information overboard--facts, principles, and knowledge cannot be dispensed with. "You cannot think in a vacuum", he says.

Recently I listened to an address by a one-time county agent, now Director of Education for a large industrial company (Mr. David Meeker). This address was titled "By our fruits"--. He drew a parallel between the products of industry and the products of education. If the products of an industrial concern are not satisfactory, something has to be done about it, something is done about it. I cannot quote him but I got the impression that he felt the students, the products of our educational institutions, are frequently unsatisfactory products but not much is done about it. As I recall it the deficiency he emphasized was their failure in communication--he called it inarticulate competence. "By our fruits" was a vigorous way to arouse the academicians.

A somewhat different way of bringing up the same matter is this statement of Director Robert D. Calkins, of the General Education Board: "Anyone who has ever had to select men for responsible positions must have been struck by the fact that there is a plethora of men with about 85% of the qualities needed and that the 15% makes the difference between satisfactory and distinguished performance." The critical 15% of unsatisfactory qualifications "whether they be in attitude, in speaking, in analytical skill, or in other capacities" constitute a responsibility which schools should but seldom do assume. The professional schools claim to select the students who are expected to have the qualifications appropriate to the profession in question, but the system of rejecting the average students and retaining and sending forth to that profession the bright students, regardless of their personal incapacities, does not insure the best professional product. I would like to raise the question whether the graduate schools are not even worse than the professional schools in this regard? Do they not admit almost wholly on the basis of high scholastic standing and confer Ph.D. degrees on the same basis, whatever the personal incapacities? If we cannot step up the academic improvement of those who are personally qualified then should we not go all out to overcome the damaging deficiencies of the academically qualified people? Throughout the entire educational program, undergraduate and graduate, we should do a better job of developing in our students those qualifications that are important to their professions. "Do Plant Pathologists need Education?" Yes, so long as there is inarticulate competence, or unadaptable competence, or uninhibited or unproductive competence--if I may be permitted to expand Mr. Meeker's neat expression.

This might well be the place to stop. My topic was in the form of a question and an answer has been given. I might well leave to the others the solution of the problem of how to make possible the education which plant pathologists need. Before stopping I must make some suggestions.

First, we need better teachers and better teaching. Our own Professor Whetzel in a paper in the Scientific Monthly (1930) said, "Perhaps no teachers in this country give less consideration to or take less stock in pedagogical theory and practice them as do college professors. Few college or university teachers, in the biological sciences at least, have ever had special training in the theory and practice of teaching. Whether this is necessary or even desirable I am not saying. It is my opinion, however, that much of the criticism now leveled at the college student

because of disinterest in his scholastic work ought to be directed against the professors who teach him." Professor Whetzel went on to say that his experience as a student and professor convinced him that the average professor had little "method in his madness," and he marveled that "we who worship so devoutly at the shrine of scientific method should practice it so little in our teaching." If we are inclined to do something about better teaching let us hope that we may proceed scientifically by subjecting new hypotheses to evaluation tests so that our solutions may be founded on facts rather than speculation. Let us be thankful that the number of those who believe that good college teaching is concerned with how as well as with what is increasing. We hear it said that teachers are born, not made. I believe they are both born and made.

In addition, we must provide programs, both at the undergraduate and graduate levels, which will contribute to the social as well as the professional or vocational effectiveness of our students. To fail to develop vocational effectiveness sells the student short--to fail to develop social effectiveness sells both the student and society short. As already said, what has been wrong is not so much over-emphasis of the vocational as under-emphasis of the humanistic side. It is not going to be easy to correct the situation but the results will surely justify the efforts. Merely to send our students for courses labelled general education is not the answer. Courses should be provided by the general education departments and they should be taken by our vocational students, but those courses (and our vocational courses as well) need more careful consideration than in the past both as to content and method, if we are to produce individuals who are personally and socially as well as professionally competent. Merely to load the student with facts about humanistic affairs doesn't get results. We must advance the attitudes as well as the facts that make for better competence and must stimulate a sense of responsibility for dealing with the humanistic problems of life. Both teacher and student must realize that what is important at graduation is not so much immediate knowledge but rather the ability to acquire the knowledge that he needs at the time when he needs it, and above all a spirit that impels him to the acquiring.

And now I am going to make a suggestion to this Society dealing with possiblities of its helpfulness in promoting plant pathology education. My attention has been called to the fact that in the engineering world there is an Engineers' Council for Professional Development. I am indebted to Dean H. P. Hammond, of our School of Engineering at Penn State, for an account of the activities of this Council. They assume the responsibility of aiding the young engineer in his professional development during the formative period of his career-the ten years that extend from the middle of high school to the end of his "internship" in industry. According to Dean Hammond this Council "has established means of aiding the high school boy to choose his profession; it has set up criteria of quality of engineering education, it has formulated canons of ethical professional practice; and it has fostered means of aiding the young engineer to promote his own advancement and to form his concepts of the place and function of the engineering profession in the national social order as well as his own place in it." These are certainly most important undertakings. In a relatively short time the Council has accomplished much through purely voluntary efforts of its members. The engineers believe it is not sufficient to have this movement resting wholly in the hands of a voluntary committee whose members are busy with their regular occupations and are planning for the services of a competent leader who can devote his full time to these broad and important responsibilities. As a national society should we not take a leaf from the engineer's book and constitute an agency for promoting the professional growth and development of young plant pathologists? The title of this paper is a question--it is fitting to raise a question in closing.

Plant pathologists need education for the service they can render public welfare and conversely there is a need for public education concerning the role of plant pathology. We must be willing to accept the challenge.

I have much satisfaction and high hopes in opening this symposium on "The Teaching of Plant Pathology."

PENNSYLVANIA STATE COLLEGE

OUTLINE OF A NEW APPROACH IN TEACHING PLANT PATHOLOGY[1]

George L. McNew

Plant pathology should be taught so as to provide the student with an opportunity to develop skill and ability for solving plant disease problems. The student must be encouraged to use his own reasoning ability and creative imagination. Any attempt to force him to memorize isolated facts about each disease is foredoomed to failure because reasoning ability seldom is developed by memorization. The student who has done nothing more than memorize a few details regarding fifty or sixty diseases is poorly qualified to go forth as a professional pathologist. Unless he has a pattern or scheme by which each of the diseases fit into the general picture of plant pathology, he will be a ready prey to hopeless confusion and disgust as soon as he encounters other diseases.

If the student is to use his creative ability in the classroom, and afterwards, he must be introduced to a logical, comprehensive system. He should find this system so natural that thousands of facts pertaining to mode of invasion, overwintering, reproduction of the pathogen, instigation of disease symptoms and control fit into a workable relationship. He should have these facts so well coordinated in his mind they will be at his ready command when he wishes to solve a problem. Instead of remembering details he can deduce with the help of the system what is most likely to prevail under a given set of conditions.

The student who is introduced to such a method of learning should find the course more inspiring. He should graduate from it with full appreciation of its potentialities and importance. If he elects to do advanced work he will be very eligible to do research because his mind will have been oriented to fundamental considerations rather than to superficial trivialities.

It is not difficult to imagine what a boon such a logical system has been to chemistry. What sort of confusion would the young chemist endure if he did not have Mendeleyev's periodic table of the elements? The chart provides nice, orderly arranged pegs upon which to pin a multitude of facts. It brings fundamental relationships into sharp focus so that the student can predict the answers to thousands of questions. He is relieved of laborious memorizing and left free for more creative intellectual pursuits.

Where is the plant pathologist to find such a system for plant diseases? Obviously it will not be easy because so many imponderables are involved in every group of diseases. There are a multitude of host plants, thousands of causal agents and many different types of host reactions. A plant disease is the compromise between two opposing sets of biological forces as refereed by environment. Heretofore, plant pathology teaching has been organized on the basis of one or the other of the three components: the host, the cause, or the disease expression. Do any of these meet the needs of a modern course by providing a desirable system?

Teaching Systems Organized by Crops or Parasites

Plant pathology cannot be organized on a fundamental basis by crops. The complexities of the subject are further emphasized and logical relationships are completely obscured. For example, the soft rot caused by Rhizopus nigricans would be taught as separate diseases of sweet potato, strawberries, asparagus, etc. Such thinking eventually leads research into the pitfalls of 1910-1915 when every bacterial soft rot of vegetables had a different species of Erwinia named as the causal agent! If plant pathology must be taught by crops, a more fundamental organization should be employed for each crop. The only justification for using the crop basis is to provide service for narrow-minded groups whose advisers are seeking an early rut for them to fall into.

The classification of plant pathology subject matter by causative agents has much to commend it; but it is very inadequate. There are, for example, certain fundamental differences in the diseases caused by the Peronosporales and Ustilaginales. However, the mycological system under which plant pathology was spawned and nourished is based on differences in sexual behavior rather than functional ability in parasitism. The formation of an appresorium, for example, may be more important in determining the type of host invasion and pathogenesis than the type of sexual reproduction. As a matter of fact, in some groups such as the Ascomycetes, the perfect stage has little to do with pathogenicity and has been completely lost by some disease fungi. More important than sex are such things as the enzyme complex and toxin production. These functional abilities determine pathogenicity.

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[1] A paper presented in the Symposium on The Teaching of Plant Pathology, Fortieth Annual Meeting of The American Phytopathological Society, Pittsburgh, Pennsylvania, December 6, 1948.

One arrives reluctantly at the conclusion that the mycological system does not fit the need of plant pathology. It is time the plant pathologist let go of the mycologists' apron strings. Instead of turning back to the mother science for orientation, they should reach out for a new science of their own -- one based on host-parasite relations. Once this step is taken the mycological considerations will revert to the mycologists who probably will do a better job of studying morphology and habitat than the pathologists. The pathologist then can develop skills in determining the nature of parasitism. Once these facts are well understood he will be in a position to work out effective control measures -- his primary professional responsibility.

The fundamental failure in systems based on either the type of host or type of causal agent is that only one member of the complex involved in a disease is given consideration. Some authors have tried to avoid using these half-truths by using a classification based upon symptoms This is of great aid in teaching identification but it is strictly superficial. Symptoms are regulated by environment so that they are seldom identical in two different localities. A root rot may cause symptoms almost identical to a wilt. The same pathogen may cause a leaf blight and a stem canker. It is obvious that some more fundamental system is needed than one based on disease expression.

A System Based upon the Physiology of Parasitism

In the final analysis plant pathology is concerned with host-parasite relations. The physiology of parasitism, therefore, becomes a major consideration. Because of this, the possibilities of using a system based on physiology was explored. The physiology of pathogens cannot be used in our present state of knowledge because we know so little about the fundamental activities that promote pathogenesis. We do, however, understand many of the host's fundamental functions. The student in plant pathology should have completed courses in general botany and plant physiology so he will have a sound foundation upon which to build if pathology is taught from a viewpoint of host physiology.

The student can be introduced to plant pathology by pointing out how important photosynthesis is to man. Our agriculture and our civilization are based upon this one chemical reaction and everything possible must be done to foster it and improve its efficiency. There is no justification for handicapping the process or destroying its products. There are many agents other than man who also depend upon the products of this photochemical process. There are many parasites that attempt to profit from destroying this food at one or more stages in the plant's development.

Chronologically, a plant may be considered as having the seven following vital functions revolving around food:

(1) Food is stored in the seed, roots, or modified stems where it maintains respiration of a bud or germ and supports growth after dormancy.
(2) The stored foods are hydrolyzed into simple sugars and amino acids, and are transferred to the expanding germ or bud in the spring.
(3) The new plant develops roots with limited food reserves that acquire water and nutrients from the soil.
(4) The food reserves from the seed and solutes from the soil are used to inaugurate meristematic activity in the young plant. The process continues throughout the plant's development with cellular growth and differentiation predetermined by genetical factors. Food materials are secured from leaves rather than seeds after the seedling stage.
(5) As the seedling enlarges water must be transferred from roots to leaves if photosynethetic activity is to be sustained at efficient levels.
(6) The leaves combine water and carbon dioxide and fix photochemical energy in the photosynthate.
(7) The simple foods must be translocated from the leaves to meristems and storage organs where they promote growth, are used in reproduction, or are stored.

The interference with any one of these seven essential functions destroys the plant's efficiency; and, if sufficiently drastic, will prevent its growth and reproduction. Such interference produces disease. A disease is, therefore, any functional disorder of a plant that prevents the synthesis, translocation, storage or efficient and economical utilization of foods. The student is introduced to plant pathology with this functional concept of disease rather than with a description of what it looks like or with a catalog of the causal agents. This immediately orients his thinking to the functional aspects rather than to memorizing descriptions and names.

When students are led to see that there is a group of diseases that interfere with each of the seven vital functions listed above, they have a system by which they can organize their learning. The system would include the following groups:

(1) Diseases caused by destruction of stored foods and storage organs. These are the soft rots and seed decays.
(2) Diseases caused by destroying seedlings before food reserves are utilized for growth. These are damping-off and seedling blight diseases.
(3) Diseases caused by preventing uptake of solutes and water from the soil. These are root rots and girdling of underground stems.
(4) Diseases caused by instigation of abnormal meristematic activity and prevention of differentiation. These are galls, club root and smuts.
(5) Diseases that prevent water movement through the xylem in plants. These are wilt diseases.
(6) Diseases that prevent photosynthesis by destroying food manufacturing tissues. Since many floral tissues and the stems are comparable to leaf tissue in morphological derivation, many of the leaf invaders cause floral blight and stem cankers as well as leaf blight, anthracnose, downy mildew, powdery mildew and rusts.
(7) Diseases that interfere with translocation of food materials. These are caused by injury to the phloem or by increasing the viscosity of phloem contents. The viruses have such effect on plants.

The Orderly Arrangement in the Proposed System

This classification of plant diseases is based upon the fundamentals of host-pathogen relationships. Such a fundamental approach would be expected to create an orderly arrangement, if it approaches a natural system. It exceeds all expectations in creating such order. The following considerations are more than indicative of a sound logical relationship.

The pathogens are arranged in order of increasing specialization. With very few exceptions, the sequence progresses from air- and soil-borne saprophytes that invade dormant food reserves through fresh wounds (class 1), through faculatitive soil saprophytes that lodge in wounds in juvenile cortical tissue (class 2), similar facultative saprophytes that invade cortical tissues of all ages through wounds or occasionally by direct penetration (class 3), facultative parasites (few obligate ones) that invade epidermal cells directly or through fresh wounds and establish themselves in cortical cells where they regulate and control cell activities rather than cause necrosis (class 4), facultative parasites specialized for xylem tissue (class 5) to those that cause a variety of leaf blights and cankers in which the parasite is intimately associated with the host cell and is most successful when it does not cause general necrosis. Many of the pathogens in classes 6 and 7 are obligate parasites or viruses that live and multiply only in the presence of living cells. They often are specialized into definite physiological races.

Some authorities advocate that perfect parasitism is to be found only in mutually beneficial symbiosis as in the mycorrhiza and legume nodule bacteria. Under such conditions both host and invader thrive. This concept implies that pathogenesis is only a by-product of parasitism caused by the ineptitude of the parasite. It is obvious in the above series that the parasites in the various classes progress away from crude invaders that cause general necrosis toward those specialized in maintaining intimate relations with the host cells.

The habitat requirements of the causal agents become progressively narrower as we progress through the seven classes. Almost any saprophyte can cause a soft rot if it falls into an open wound on a moist reservoir of stored food. Relatively fewer invade the growing root tissue and cause necrosis and most of the wilt and gall producers are well specialized. At the other end of the scale the pathogens are specialized for one specific type of host cell and any small change is fatal to them. Usually a single gene difference is enough to render the cell immune to them.

Disease control measures may be grouped according to the classes of disease. In the three lower classes, control is effected by modifying the environment and only rarely by breeding for resistance. In classes 4 and 6 an attempt must be made to avoid or restrict the parasite by sanitation, rotation or spraying. Beginning with class 4, breeding for disease resistance becomes increasingly important until it is the principal measure in classes 6 and 7 (rusts and viruses).

Types of Parasites Assembled in the Various Groups

The system brings together a number of diseases that may strike the mycologically trained person as being totally unrelated. Some of these might be examined more minutely from the viewpoint of the pathologist.

In class 1, the soft rots caused by Rhizopus nigricans (sweet potato) and Erwinia carotovora

(carrots) are discussed in the same lecture. And, why not? Both are caused by saprophytes that happen to land in an open wound. Their pathogenic abilities hinge upon the production of protopectinase, pectinase and amylase. Both are controlled by proper storage and avoiding wounds in harvest.

The discussion of seed decay in cold soils leads the student to damping-off (class 2). In class 2, onion smut and seedling blight are discussed jointly. Why should a student study them some twelve weeks apart as is done in the mycological system because one is caused by a Phycomycete (imperfect, Ascomycete or Basidiomycete) and the other by a Basidiomycete? Both diseases are caused by soil-inhabiting fungi that produce mycelium capable of penetrating only juvenile tissue. They are most destructive in cold soils where the host is retained in a juvenile state for a prolonged period. Both are controlled by local soil sterilization ordinarily attained by use of seed treatments. In many areas the only difference in control is in dosage requirement.

In class 3 such diseases as root rots of corn (Pythium), cotton (Phymatotrichum) and tobacco (Thielaviopsis) are discussed jointly. In class 4 such diseases as club root and crown gall are discussed as a unit. They are both soil inhabitants that invade juvenile or wounded cells, and regulate cellular activity within the host. Control is by sanitation and crop rotation. The smut diseases fall in this class even though they grow throughout the vegetative tissues without causing hyperplasia or necrosis and instigate abnormal development only when they invade buds or floral tissues. Most of the smut organisms invade undifferentiated seedling tissues and exist intracellularly the same as Plasmodiophora.

Subdivisions should naturally be made in class 5 according to the habit of the wilt parasites: (a) soil-borne (Fusarium and Verticillium), (b) insect-transmitted (Graphium, Bacterium) and (c) contact-transmitted bacteria that penetrate through hydathodes and stomata.

In class 6, the subdivisions should be made according to the specialization of the parasitism: (a) leaf blights and stem cankers, (b) downy mildew, (c) powdery mildew, (d) autoecious rusts and (e) heteroecious rusts. The system follows a mycological approach fairly closely except in the first subdivision. Therein diseases such as anthracnose and bacterial blights of beans will be discussed in the same lecture. It is logical since they overwinter in the seed, reach the trifoliate leaves through the same medium and are controlled by a two year crop rotation and use of western grown seed.

All of the viruses are discussed in class 7 but it is obvious that as soon as more is known about their physiology some probably will be placed in classes 4 and 6.

Summary

A fairly natural system for studying plant pathology may be devised by classifying diseases according to their interference with fundamental activities of the host in synthesizing and utilizing food materials. Seven classes are recognized based upon: (1) destruction of food reserves, (2) prevention of establishment of seedlings, (3) interference with procurement of water and nutrients from the soil, (4) stimulation of abnormal meristematic activity that will dissipate food reserves, (5) prevention of water movement to the leaves, (6) destruction of photosynthetic ability, (7) interference with translocation of foods. A study of the pathogens falling in each of these classes shows that they have automatically been arranged in a surprisingly orderly fashion ranging from:

(1) Soil and air saprophytes through facultative parasites to obligate parasites.
(2) Wound invaders, through stomata and lenticel invaders to direct, cell-penetrating forms.
(3) Gross destroyers of all food materials to delicately balanced parasites that maintain an intimate, balanced relationship with the host cell.
(4) Those controlled by modifying the environment, through escaping or restricting the parasite, to those controlled by breeding.

The system is a natural one to the extent that it places the various diseases in a logical, orderly arrangement so the student will not be forced to memorize every detail. He will automatically know that a disease of class I is probably a wound invading saprophyte controlled by changing the environment while a highly specialized rust parasite of class VI may have an alternate host and be specialized into many physiologic races which can be controlled by breeding. The student's mind is directed toward the fundamental relations and relieved of memory work so he can exercise his creative ingenuity and develop enthusiasm for phytopathology.

The system is far from perfect but the relationships are so logical that they warrant further examination and trial by instructors of phytopathology. Some of the inconsistencies will undoubt-

edly be clarified as more data accumulate on host-parasite relations and as studies on the physiology of parasitism are undertaken.

FORMERLY AT IOWA STATE COLLEGE, AMES, IOWA, NOW AT BOYCE THOMPSON INSTITUTE, YONKERS, NEW YORK

SHOULD A STANDARD UNDERGRADUATE COURSE IN PLANT PATHOLOGY BE FORMULATED?[1]

S. G. Lehman

The very great significance of plant diseases as related to the well-being of mankind indicates the urgent need for effective instruction regarding their cause, effects, and control. One very important place for such instruction is in the undergraduate or introductory course in plant pathology.

The teacher's conception of the aim or purpose of the undergraduate course will to a considerable extent determine his choice of subject matter, the order of presentation, and the emphasis he will give to different aspects of the subject matter. In this symposium, the matters of aim, content, method of presentation and teacher preparation will be discussed by other participants. Regarding these questions only the following very brief statement is made here.

A standard undergraduate course in plant pathology should consist of a series of lectures, recitation and laboratory periods dealing with a minimum number of plant diseases. Some of these should be representative of each type of disease and adequate to serve as a sufficient example to acquaint the student with the nature, behavior, and effects of all important kinds of plant pathogens. The instruction should point to a high level of scientific achievement in the solution of plant disease problems, should indicate problems still unsolved and should seek to interest not only students of agriculture, but students in all fields of biologic endeavor.

If teachers of plant pathology can agree on the need for a standard prospectus for the undergraduate course presumably one will be formulated and put into print and thus made available to all teachers. Will the effectiveness of teaching in plant pathology be increased by formulation of a standard procedure for undergraduate instruction?

Certain arguments may be offered against the formulation of a standard undergraduate course. There is some doubt that a standard course will be received favorably by many teachers. Why give time and effort to the problem of defining a standard, if, as some believe, few teachers will submit to be guided by it to any significant degree? According to this view, a goodly proportion of the persons presently teaching plant pathology are individualistic and nonconformist in attitude, especially towards questions of educational procedure. Firmly convinced that their choice of subject matter and manner of presentation is best, these persons may refuse to accept such suggestions of change as would be implied in the formula for a standard course. Of course, the opinions of all experienced teachers should be given due consideration in any attempt at writing a standard prescription for the undergraduate course in plant pathology. But, when the teaching practice currently in vogue differs from such standard prescription as may be set up, there is some question of the extent to which current practice will be modified voluntarily to meet the prescribed standard.

The formulation and official endorsement of a standard undergraduate course for plant pathology might conceivably act as an impediment to good teaching. Particularly is this true if the range of subject matter is specified within too narrow limits and the method of presentation is prescribed too exactly. The teacher might find it difficult or quite impossible to obtain suitable specimen material if the diseases prescribed for study do not occur in the area where he works. Moreover a student develops a deeper and more impelling interest in plant pathology if the examples of the different types of diseases selected for study are drawn from crops with which he has had some intimate experience. If his contact with potatoes, let us say, is limited to the few tubers he may have seen in the grocery store, a study of late blight may seem pretty dull and inconsequential. If he has worked in a potato field, however, and a considerable part of his family's income has come from the culture of potato, a study of late blight of potato will easily command his interest. On the other hand, a standard course which prescribed a study of potato diseases and excluded the use of diseases of some other crop, say tobacco, as examples of disease types with students drawn from an area where agronomic and economic interest centers in tobacco to the virtual exclusion of potato would place an unnecessary handicap on the teaching of plant pathology. Teachers of plant pathology should be free to take advantage of all interests inherent and natural in the community from which their students come.

A number of considerations would seem to indicate the need for formulation of a standard undergraduate course in plant pathology. The existence of a recognized standard for this course

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[1]A paper presented in the Symposium on The Teaching of Plant Pathology, Fortieth Annual Meeting of The American Phytopathological Society, Pittsburgh, Pennsylvania, December 6, 1948.

would be a very considerable aid to the person just beginning his teaching career. After completion of advanced studies leading to an M. S. or Ph.D. degree, and sometimes immediately after receipt of his undergraduate degree, the graduate accepts a position where at least part of his duties is to give instruction in plant pathology. The advanced graduate with a major in botany may have had no course or only an introductory course in plant pathology. Again the student who majors in some aspect of applied botany as farm crops, fruits, vegetables, or genetics may have had as little as a single introductory course in plant pathology. If, as may happen, such graduate finds himself in a new position where he is to give lectures or direct laboratory work in plant pathology, is he properly equipped for the task? Does he know what to present in order to achieve the intent and purpose of teaching plant pathology? He is likely to need advice or suggestion in selecting specific diseases for class study and in determining what and how much to present and what and how much to leave for the student to dig out for himself. A formulated standard for the undergraduate course could serve as a helpful guide to the beginning teacher with limited background and experience.

Perhaps the majority of persons who undertake the teaching of courses in plant pathology in the future will be advanced graduates who have completed majors involving several courses at the undergraduate and graduate levels. Presumably such students will have a fairly adequate conception of the aims of instruction in plant pathology. However, many students who earn Ph.D.'s with majors in plant pathology, have little or no experience in teaching the courses in that field. Many of them make their entire contribution toward the activity of the department with which they are associated by helping in the field of research. Through their assistance in research, much has been added to the total of knowledge in plant pathology, but they have gained little or no experience in classroom teaching. These new teachers will welcome such guidance as might be obtained from a formulated standard course.

In some schools, the undergraduate course is in content and method based largely on the experience of one person, the present teacher of the course. If this person is actively participating in research, he may more or less unwittingly come to place undue emphasis on the diseases which are objects of his research and give only meager attention to others of equal or greater merit. The greater portion of his illustrative material will be drawn from the field of his research interest, fruit diseases, or cereal diseases, or vegetable diseases, or even from diseases of a single crop as tobacco. Thus his students may come short of gaining a comprehensive view of the whole problem of plant diseases. Such a teacher, experienced though he may be, gives his students a very restricted conception of the diverse nature and relative importance of plant diseases. Presumably if a standard course ever is set up, its formulation will be by a group of persons who have had some years of experience in research and teaching. Being the product of the experience of persons working each in his own restricted field of research interest but together covering the whole gamut of plant disease problems, this formulation should serve as a reservoir of valuable suggestions even to the experienced teacher who is earnestly seeking to give his students a sufficiently comprehensive and intensive view to motivate them favorably toward the science of plant pathology.

In the course of appraising the abilities of applicants for scholarships and fellowships in our graduate schools, it is customary to require a transcript showing the credits, grades, and points earned in undergraduate courses. By these marks, we appraise, in some measure, the ability of the applicant to do advanced work. However, we are often reluctant to accept these grades as having much significance because of a number of factors. One of these is a lack of knowledge of the type and content of the course for which the grades were awarded. Was the subject matter presented sufficiently difficult to serve as a real test of the student's ability to progress in learning? Obviously, the adoption and use of a recognized standard course of instruction at the undergraduate level would remove one of the elements of uncertainty in estimating an applicant's ability on the basis of the grade given him. Furthermore, adoption of the standard course should remove the necessity for the often imposed requirement that the student repeat his introductory course when enrolling for advanced work in another school.

Another advantage in favor of formulation of a standard undergraduate course will be found in the matter of providing teaching aids helpful in creating and maintaining interest. Seldom is any one teacher able to prepare by his own labor good lantern slides illustrative of all aspects of the diseases he wishes to discuss. If all teachers will offer their best lantern slides for duplication, complete sets can be prepared to fortify each lecture or illustrate each phase of the subject matter. These sets could be made available to all teachers at minimum cost. Other teaching aids such as microscope slides, wall charts, laboratory outlines, quiz sheets and diagrams could be prepared to fit a standard course.

As a rule formal instruction in plant pathology is given only at the college level. Only a small proportion of college students receive this instruction. The standard formulated for the

introductory course might conceivably be adapted to use in high schools and in schools of vocational agriculture. Many students in vocational agriculture never enter college, but return to agricultural pursuits without having instruction in plant pathology. The tremendous impact of plant diseases on human welfare would seem to indicate that a larger part of our total student population should be receiving instruction in this science. Only when a large segment of the public have an adequate understanding of the nature of plant diseases and how they affect our economy will plant pathology receive the recognition it deserves.

With many of the now too few students who take courses in plant pathology, the undergraduate course will be both the first and the last course taken. Some greater measure of understanding than at present exists as to the principles to be emphasized, the kind and quantity of subject matter required to fix the important ideas, and the most effective method of presentation of the subject matter is highly desirable for this course. The publication of a standard formula for this course is desirable as a valuable guide for young instructors and a source of helpful suggestions to experienced teachers in their immediate efforts to interest the student and in their ultimate aim to promote public welfare through instruction in plant pathology.

NORTH CAROLINA STATE COLLEGE OF AGRICULTURE AND ENGINEERING, RALEIGH

THE CONTENT OF A GENERAL COURSE IN PLANT PATHOLOGY[1]

L. D. Leach

The material that should be included in an undergraduate course in Plant Pathology is an important concern of the instructor in charge. Circumstances are so variable, however, that it is probably neither feasible nor desirable to present identical courses in the numerous colleges and universities presenting instruction in this subject.

For this reason, I would hesitate to suggest the material that should be included by instructors in other schools or areas, but I am glad of the opportunity to discuss the subject matter included in our beginning course at Davis and the reasons for the selection.

In my opinion the content of a course should be determined by the needs of the student and by the probable use that the students will make of the subject matter after their graduation. With this in mind, we first analyze our enrollment and consider the type of student with which we deal.

Students in the four year curriculum at Davis are required to have a B average in 12 recommending units from high school and therefore, represent only the upper 10 to 20 percent of high school graduates. Others may transfer from Junior College after they demonstrate scholastic ability by maintaining a C+ average. Students normally take Plant Pathology 120 during their junior year after strong basic training in Botany, Zoology, Chemistry and Bacteriology and are therefore capable of understanding technical information.

An analysis of the class enrollment shows that 80 to 85 percent of our students are majors in plant science with special interests in Pomology, Viticulture and Enology, Floriculture, Truck Crops and Agronomy. About 10 to 15 percent are majoring in Agricultural Education. Of the total number, about 10 percent are graduate students and 10 to 15 percent are from foreign countries. Usually not more than 5 percent are preparing for graduate work in Plant Pathology.

After graduation, we find that 37 percent of our Plant Science and Agricultural Education majors accept employment as High School Vocational Agricultural Teachers, 25 percent engage in farming operations, 8 percent are employed in Agricultural Extension activities, 10 percent are employed in commercial agriculture, while 12 percent engage in teaching or research on the college level or are employed by State or Federal agencies. As time after graduation elapses the number engaged in high school teaching decreases with a corresponding increase in farming and Agricultural Extension.

With this class composition in mind, it is evident that the course should be designed to meet the needs of students who will use their knowledge and experience in plant pathology as it applies to farming practice either as teachers, farm advisors, ranch managers or in agricultural business. To emphasize material of primary interest to the few majors in plant pathology would not be justifiable.

The broad objectives of the course as viewed by the instructor are:

First - To stimulate interest in the subject by bringing to the students' attentions some of the outstanding examples of serious plant disease outbreaks or outstanding examples of spectacular control of plant diseases. Whenever an opportunity presents itself we dramatize the important economic aspects and human relations of plant diseases as a means of stimulating and maintaining interest.

Second - To convince the student of the practical aspects of a knowledge of plant diseases particularly as it may affect his future occupation or agricultural production.

Third - Having attracted his attention and interest, we hope to show that a knowledge of the life cycle of the causal agent, of pathological anatomy and physiology, and of the relation of environment and cultural practices is necessary to an understanding of disease development and control. If we accomplish this objective, then we will have added to his store of basic information and increased his ability to exercise discretion in the acceptance of statements or claims.

Fourth - To familiarize the student with the language of the science and with sources of information so that he can locate and understand publications on plant diseases whenever a need arises.

We realize that the student will soon forget much of the factual information in the course but we hope that whenever the need for information on plant diseases develops he will know where to find the information and will be able to understand and to apply the information when it is con-

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[1]A paper presented in the Symposium on The Teaching of Plant Pathology, Fortieth Annual Meeting of The American Phytopathological Society, Pittsburgh, Pennsylvania, December 6, 1948.

veyed to him.

The course as presented consists of two one-hour lectures and two three-hour laboratories. Frankly we believe that most of our effective teaching is done in the laboratory periods. Lectures are used primarily to summarize and amplify the laboratory material, to present an overall picture of plant pathology and to stimulate interest. Portions of the lecture periods are utilized for quizes and examinations since the authorities insist that we assign a measure of accomplishment to each student at the end of the semester.

The laboratory topics are selected to represent the diseases of major economic importance upon California crops and include examples from deciduous and citrus fruits, truck and field crops as well as ornamentals and forest trees. They are also selected to include diseases caused by fungi, bacteria, viruses and nematodes. About 12 laboratory periods are devoted to a detailed study of 12 specific diseases such as Brown Rot of Stone Fruits, Bacterial Canker of Tomatoes and Curly Top of Sugar Beets. Two or three laboratory periods are devoted to each of 6 more general topics such as Seedling Diseases and their control, Downy Mildews, Powdery Mildews, Smuts, Rusts and Virus Diseases. For example in the case of smut diseases the first period is devoted to a detailed study of one or two smuts while the second period is spent on a comparison of life cycles and control of a number of additional examples.

Two periods are reserved for field trips during which diseased plants are observed in their natural surroundings. After they have completed the study of deciduous fruit diseases one period is devoted to preparation and comparison of spray fungicides.

We find it important to devote the first two laboratory periods to a general introduction. First we make sure that every student is familiar with the correct illumination and use of the microscope using prepared slides of disease specimens. Next we introduce the subject of seedling disease not as a specific disease but as a complex involving several crop plants, and three or four different fungi. Emphasis is placed upon pathogenicity, upon the relation of the fungus to host tissue, the effect of the environment and the use of protective fungicides.

Throughout the course an attempt is made to provide healthy and diseased specimens for each laboratory. This is accomplished by growing and inoculating plants in the greenhouse, by collections from field plots and orchards maintained for this purpose and by collections of diseased specimens from different parts of the state during field trips.

This is possible because the climate in different areas of the state permits the production of a variety of winter crops. Even so, it is necessary to arrange our schedule to coincide with available material rather than following a taxonomic arrangement. To provide fresh materials requires considerable time and effort but we believe it is justified by the improvements in student attitude.

Our primary objective in a laboratory exercise is to aid the student in discovering the interrelations between the causal factors and the host. He is first provided with specimens of healthy and of diseased plants showing typical symptoms. A mimeographed outline is also furnished containing suggestions for procedure in the study of the specific disease with enough information to aid the student in reaching the proper conclusions and with a list of pertinent references.

Students work either individually or in groups of two or three. They are encouraged to make their own observations and to reach independent conclusions. Considerable emphasis is placed upon pathological anatomy and the students find some familiarity with normal plant anatomy a distinct advantage.

In my opinion all of our textbooks have underemphasized the importance of pathological anatomy and physiology in the understanding of plant disease. The few examples where fairly complete information is available, such as Dr. Esau's and Dr. Bennett's papers on pathological anatomy and physiology of curly top of sugar beets and Dr. Larsen's paper on normal and pathological anatomy of cabbage affected by club root, are extremely valuable for teaching purposes.

At the appropriate time in our laboratory exercise prepared slides are provided to show details not discernible in free hand sections. These observations usually lead directly to the relation of the causal organism in the case of parasitic diseases. With fungus diseases pure cultures of the organism in different stages of development are provided as well as prepared slides and mounts of germinating spores.

After the student is familiar with growth cycle of the host and with the complete life cycle of the causal agent he then can construct a cycle of development of the disease including the relation of cultural practices and environmental factors.

Every effort is made to convince the student that a disease is not a static condition but rather a progressive series of interactions between the host and the causal factor dependent in degree or extent upon weather conditions and upon agricultural practices. In this procedure we follow closely the published expressions of Dr. Whetzel who has contributed so much clear thinking to the teaching of Plant Pathology.

The thinking process of a student studying a laboratory exercise on a specific plant disease closely parallels the steps of a research worker in studying a new plant disease. First the students learn to recognize the symptoms of the specific disease and to distinguish infected from healthy plants, as well as from plants affected by other similar diseases. Next, by observations of free hand or prepared sections he finds a logical explanation for the external symptoms. By studying all stages in the life cycle of the causal agent and connecting each stage with the seasonal development of the host, a cycle of development of the disease can be constructed. Finally, by discovering one or more weak links in the cycle, the student is able to predict the types of control measures that would most likely prove successful. He is in a position, therefore, to understand the results of experimental trials and the reasons for control recommendations.

The procedures of the student and the research worker are similar except that the student with access to selected plant materials, and aided by published reports and oral suggestions, is able to cover within a three-hour laboratory period the information developed by research activities extending over several years.

In my opinion, it makes little difference whether we study white pine blister rush or cedar apple rust as an example of a heteroecious rust having its aecial stage on the economic host. In the same way it makes little difference whether we study Coryneum blight of stone fruits or apple scab as an example of a fruit disease where critical timing of fungicides is dependent upon a knowledge of the complete cycle of development and where the most obvious symptoms are the result of defensive reactions on the part of the host.

The important consideration is that students in their general course be given a clear picture of the science of plant pathology and that they leave the course with real interest in the subject and with a desire to know more about the continuing developments in the field.

UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA

UNDERGRADUATE PREPARATION FOR PLANT PATHOLOGISTS[1]

J. G. Leach

When I was invited to take part in this symposium it was suggested that I speak on the topic: "Course Requirements for an Undergraduate Major in Plant Pathology". I requested a change of topic for the simple reason that I do not think there is any place for an undergraduate major in plant pathology.

The best undergraduate training for the student who is to become a plant pathologist should include very few courses in plant pathology. All of the available time as an undergraduate should be devoted to the basic courses in biological sciences, mathematics, chemistry, physics, English, foreign languages, and the essential courses in agriculture and the social sciences. Courses in plant pathology beyond an introductory course, and perhaps a beginning course in mycology, should be left until the student is in the graduate school. Undergraduate students who take advanced or specialized courses in plant pathology usually take them at the expense of the basic courses and, because they have not had the basic courses, do not get as much from the plant pathology subjects as they should. Also, because the undergraduate student usually has not acquired a professional outlook, he misses much of the value of the plant pathology subjects. Then when he becomes a graduate student he will have to take the same courses over or go back and take the basic undergraduate courses without graduate credit; neither of which is very satisfactory. It may inflate our professional pride to have in the college catalog a plant pathology major, but I can see no other justification for it.

If there is to be no plant pathology major, what kind of an undergraduate curriculum should our future plant pathologist follow? First let us consider the situation as it actually is in most institutions from which graduate students in plant pathology are drawn. Students who eventually become plant pathologists usually come up through either the Agricultural college or the Liberal Arts college. Few of the curricula usually open to the students in these colleges provide ideal preparation for the student who is to major in Plant Pathology in the graduate school.

In the college of agriculture one usually can specialize in various phases of agronomy, horticulture, animal husbandry, dairy husbandry, agricultural engineering, vocational agriculture or in the so called general course which is supposed to prepare him to be a practical farmer or county agent. The curricula in agronomy or horticulture are usually so filled with the multiplicity of specialized courses in the major department that time is not available for adequate courses in botany, chemistry, mathematics, English or foreign languages. It is safe to say that few graduates of our colleges of agriculture are adequately prepared for graduate work in plant pathology or any other plant or animal science.

The student who comes up through the Liberal Arts college is usually better prepared in these basic subjects but is often completely lacking in farm experience and the agricultural viewpoint which is so desirable for most fields of plant pathology.

What then is the solution of this problem? I have two suggestions to offer. First: Every College of agriculture should have a curriculum in agriculture science or science specialization in which those students who plan to take graduate work in plant pathology or other plant sciences can omit a few courses in stock judging and apple polishing and take more of the basic courses not included in the so called "professional" curricula. I do not know how many agricultural colleges have such a curriculum at the present time but about 8 years ago I examined the curricula of every Land Grant college in the country and found only four. But even if such a curriculum were available in every agricultural college the problem would be only partly solved. Unfortunately, most of our undergraduate students do not know that they are going to take graduate work until they have completed or almost completed their undergraduate work (most likely in the wrong curriculum). The problem is to discover the potential graduate student early in his college career and guide him into the science specialization curriculum as soon as possible. This calls for an adequate undergraduate advisory service and a clear understanding of the situation that I have just outlined to you by those who advise the students. Much will depend upon the advisors but I maintain that such a curriculum in the catalog will help the student and will make the advisors' work more effective. I am afraid that we, as teachers in the graduate school, have been too content to take without question all those who knock on our doors. We should show a greater interest in our graduate students while they are still freshman. Does your college catalog have a science specialization curriculum in it? If not why not get one put in?

WEST VIRGINIA UNIVERSITY

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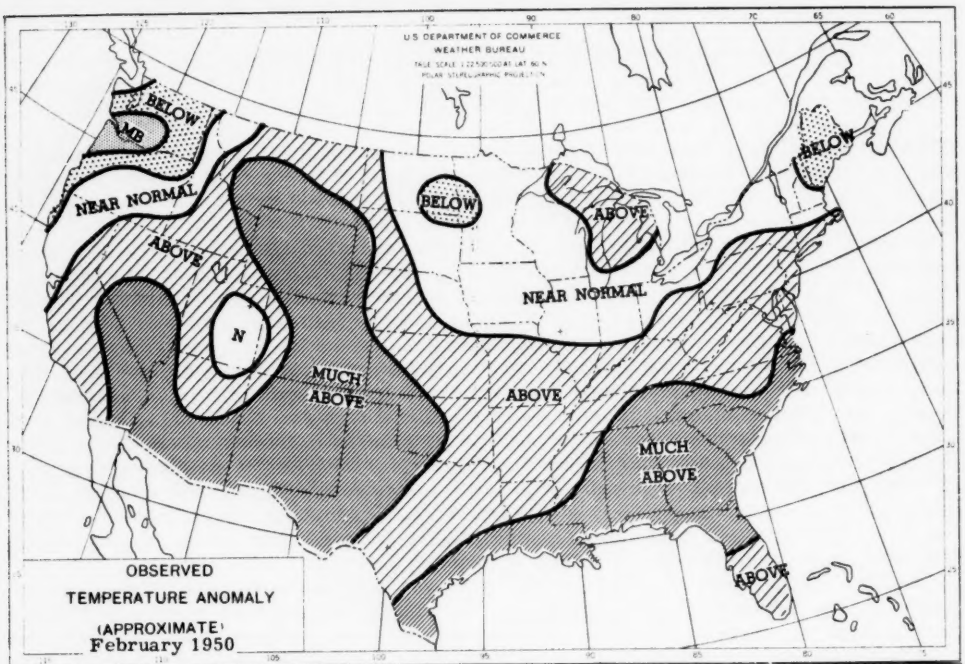
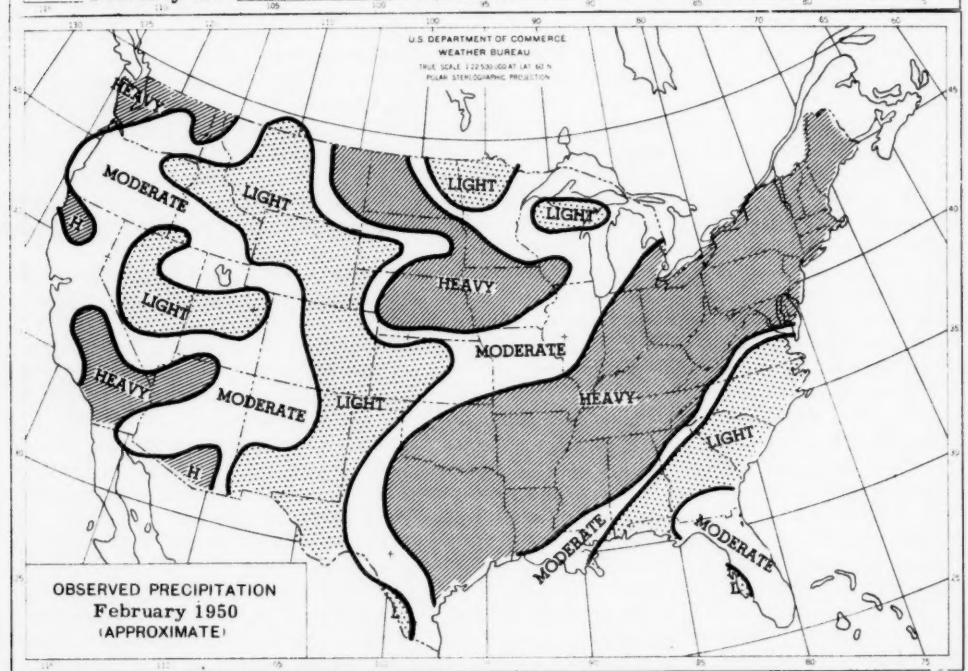
[1] A paper presented in the Symposium on The Teaching of Plant Pathology, Fortieth Annual Meeting of The American Phytopathological Society, Pittsburgh, Pennsylvania, December 6, 1948.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.